Refine Search

Search Results -

Terms	Documents
L1 and L2	0

Database:
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Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L3

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Search History

DATE: Saturday, May 13, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L3	l1 and l2	0	L3
L2	colas.in.	111	L2
L1	thioredoxin and peptide aptamer	3155	L1

END OF SEARCH HISTORY

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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 4	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 5	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 6	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 7	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 8	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 9	MAR 08	X.25 communication option no longer available after June 2006
NEWS 10	MAR 22	EMBASE is now updated on a daily basis
NEWS 11	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 12	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS 13	APR 04	STN AnaVist $500 visualization usage credit offered
NEWS 14	APR 12	LINSPEC, learning database for INSPEC, reloaded and enhanced
NEWS 15	APR 12	Improved structure highlighting in FQHIT and QHIT display in MARPAT
NEWS 16	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected
NEWS 17	MAY 10	CA/CAplus enhanced with 1900-1906 U.S. patent records
NEWS 18	MAY 11	KOREAPAT updates resume

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
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* *

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FILE 'HOME' ENTERED AT 14:46:37 ON 13 MAY 2006

=> file medline, biosis, wpids, dgene
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.21 0.21

FILE 'MEDLINE' ENTERED AT 14:46:58 ON 13 MAY 2006

FILE 'BIOSIS' ENTERED AT 14:46:58 ON 13 MAY 2006
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=> s peptide aptamer
L1 311 PEPTIDE APTAMER

=> s l1 and thioredoxin
L2 10 L1 AND THIOREDOXIN

=> d l2 ti abs ibib tot

L2 ANSWER 1 OF 10 MEDLINE on STN
TI Sequence-specific peptide aptamers, interacting with the intracellular domain of the epidermal growth factor receptor, interfere with Stat3 activation and inhibit the growth of tumor cells.
AB Receptor tyrosine kinases of the epidermal growth factor (EGF) receptor family regulate essential cellular functions such as proliferation, survival, migration, and differentiation but also play central roles in the etiology and progression of tumors. We have identified short peptide sequences from a random peptide library integrated into the **thioredoxin** scaffold protein, which specifically bind to the intracellular domain of the EGF receptor (EGFR). These molecules have the potential to selectively inhibit specific aspects of EGF receptor signaling and might become valuable as anticancer agents. Intracellular expression of the aptamer encoding gene construct KDI1 or introduction of bacterially expressed KDI1 via a protein transduction domain into EGFR-expressing cells results in KDI1.EGF receptor complex formation, a slower proliferation, and reduced soft agar colony formation. Aptamer KDI1 did not summarily block the EGF receptor tyrosine kinase activity but selectively interfered with the EGF-induced phosphorylation of the tyrosine residues 845, 1068, and 1148 as well as the phosphorylation of tyrosine 317 of p46 Shc. EGF-induced phosphorylation of Stat3 at tyrosine

705 and Stat3-dependent transactivation were also impaired. Transduction of a short synthetic **peptide aptamer** sequence not embedded into the scaffold protein resulted in the same impairment of EGF-induced Stat3 activation.

ACCESSION NUMBER: 2003440696 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12842895
TITLE: Sequence-specific peptide aptamers, interacting with the intracellular domain of the epidermal growth factor receptor, interfere with Stat3 activation and inhibit the growth of tumor cells.
AUTHOR: Buerger Claudia; Nagel-Wolfrum Kerstin; Kunz Christian; Wittig Ilka; Butz Karin; Hoppe-Seyler Felix; Groner Bernd
CORPORATE SOURCE: Georg Speyer Haus, Institute for Biomedical Research, Paul Ehrlich Strasse 42, D-60596 Frankfurt am Main, Germany.
SOURCE: The Journal of biological chemistry, (2003 Sep 26) Vol. 278, No. 39, pp. 37610-21. Electronic Publication: 2003-07-02.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200311
ENTRY DATE: Entered STN: 23 Sep 2003
Last Updated on STN: 18 Dec 2003
Entered Medline: 17 Nov 2003

L2 ANSWER 2 OF 10 MEDLINE on STN
TI Inhibition of an activated Ras protein with genetically selected peptide aptamers.
AB Mutant alleles of Ras maintain an activated, GTP-bound conformation and relay mitogenic signals that cannot be turned off. A genetic selection in Saccharomyces cerevisiae was used to identify peptide aptamers that suppress the growth arrest phenotype of an activated Ras allele. Peptide aptamers were expressed as C-terminal fusions to glutathione-S-transferase. Modifications that alter the coding capacity of the **peptide aptamer** indicate it is necessary for Ras2-Val19 suppression. Aptamer expression also reduces the elevated levels of cAMP and suppresses the heat shock sensitivity characteristic of Ras-activated yeast cells. The **peptide aptamer** retains suppressor activity when fused to **thioredoxin**. The **peptide aptamer** expression strategy described here indicates that aptamers presented as unconstrained peptides have functional capacity in vivo. Copyright 2003 Wiley Periodicals, Inc. Biotechnol Bioeng 82: 38-46, 2003.
ACCESSION NUMBER: 2003058408 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12569622
TITLE: Inhibition of an activated Ras protein with genetically selected peptide aptamers.
AUTHOR: Kurtz Stephen E; Esposito Kim; Tang Weimin; Menzel Rolf
CORPORATE SOURCE: Department of Immunology, Veterans Affairs Medical Center, Portland, Oregon 97201, USA.. skurtz@qwest.net
SOURCE: Biotechnology and bioengineering, (2003 Apr 5) Vol. 82, No. 1, pp. 38-46.
Journal code: 7502021. ISSN: 0006-3592.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200309
ENTRY DATE: Entered STN: 6 Feb 2003
Last Updated on STN: 28 Sep 2003
Entered Medline: 26 Sep 2003

L2 ANSWER 3 OF 10 MEDLINE on STN
TI Selection of genetic agents from random **peptide aptamer** expression libraries.
ACCESSION NUMBER: 2001127545 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11075346
TITLE: Selection of genetic agents from random **peptide aptamer** expression libraries.
AUTHOR: Geyer C R; Brent R
CORPORATE SOURCE: Molecular Sciences Institute, Berkeley, California 94704, USA.
SOURCE: Methods in enzymology, (2000) Vol. 328, pp. 171-208. Journal code: 0212271. ISSN: 0076-6879.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 22 Feb 2001

L2 ANSWER 4 OF 10 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Sequence-specific peptide aptamers, interacting with the intracellular domain of the epidermal growth factor receptor, interfere with Stat3 activation and inhibit the growth of tumor cells.
AB Receptor tyrosine kinases of the epidermal growth factor (EGF) receptor family regulate essential cellular functions such as proliferation, survival, migration, and differentiation but also play central roles in the etiology and progression of tumors. We have identified short peptide sequences from a random peptide library integrated into the **thioredoxin** scaffold protein, which specifically bind to the intracellular domain of the EGF receptor (EGFR). These molecules have the potential to selectively inhibit specific aspects of EGF receptor signaling and might become valuable as anticancer agents. Intracellular expression of the aptamer encoding gene construct KDI1 or introduction of bacterially expressed KDI1 via a protein transduction domain into EGFR-expressing cells results in KDI1cntdotEGF receptor complex formation, a slower proliferation, and reduced soft agar colony formation. Aptamer KDI1 did not summarily block the EGF receptor tyrosine kinase activity but selectively interfered with the EGF-induced phosphorylation of the tyrosine residues 845, 1068, and 1148 as well as the phosphorylation of tyrosine 317 of p46 Shc. EGF-induced phosphorylation of Stat3 at tyrosine 705 and Stat3-dependent transactivation were also impaired. Transduction of a short synthetic **peptide aptamer** sequence not embedded into the scaffold protein resulted in the same impairment of EGF-induced Stat3 activation.
ACCESSION NUMBER: 2003:542884 BIOSIS
DOCUMENT NUMBER: PREV200300546950
TITLE: Sequence-specific peptide aptamers, interacting with the intracellular domain of the epidermal growth factor receptor, interfere with Stat3 activation and inhibit the growth of tumor cells.
AUTHOR(S): Buerger, Claudia; Nagel-Wolfrum, Kerstin; Kunz, Christian; Wittig, Ilka; Butz, Karin; Hoppe-Seyler, Felix; Groner, Bernd [Reprint Author]
CORPORATE SOURCE: Georg Speyer Haus, Institute for Biomedical Research, Paul Ehrlich Strasse 42, D-60596, Frankfurt am Main, Germany groner@em.uni-frankfurt.de
SOURCE: Journal of Biological Chemistry, (September 26 2003) Vol. 278, No. 39, pp. 37610-37621. print. CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 19 Nov 2003
Last Updated on STN: 19 Nov 2003

L2 ANSWER 5 OF 10 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Inhibition of an activated Ras protein with genetically selected peptide aptamers.
AB Mutant alleles of Ras maintain an activated, GTP-bound conformation and relay mitogenic signals that cannot be turned off. A genetic selection in Saccharomyces cerevisiae was used to identify peptide aptamers that suppress the growth arrest phenotype of an activated Ras allele. Peptide aptamers were expressed as C-terminal fusions to glutathione-S-transferase. Modifications that alter the coding capacity of the **peptide aptamer** indicate it is necessary for Ras2-Val19 suppression. Aptamer expression also reduces the elevated levels of cAMP and suppresses the heat shock sensitivity characteristic of Ras-activated yeast cells. The **peptide aptamer** retains suppressor activity when fused to **thioredoxin**. The **peptide aptamer** expression strategy described here indicates that aptamers presented as unconstrained peptides have functional capacity in vivo.
ACCESSION NUMBER: 2003:182129 BIOSIS
DOCUMENT NUMBER: PREV200300182129
TITLE: Inhibition of an activated Ras protein with genetically selected peptide aptamers.
AUTHOR(S): Kurtz, Stephen E. [Reprint Author]; Esposito, Kim; Tang, Weimin; Menzel, Rolf
CORPORATE SOURCE: Department of Immunology, Veterans Affairs Medical Center, Portland, OR, 97201, USA
skurtz@qwest.net
SOURCE: Biotechnology and Bioengineering, (April 5 2003) Vol. 82, No. 1, pp. 38-46. print.
CODEN: BIBIAU. ISSN: 0006-3592.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Apr 2003
Last Updated on STN: 9 Apr 2003

L2 ANSWER 6 OF 10 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Phosphatidylinositol 3-kinase and Ras: Searching for peptide aptamers.
ACCESSION NUMBER: 2002:411383 BIOSIS
DOCUMENT NUMBER: PREV200200411383
TITLE: Phosphatidylinositol 3-kinase and Ras: Searching for peptide aptamers.
AUTHOR(S): Choy, C. P. [Reprint author]
CORPORATE SOURCE: Plano Senior High School, Plano, TX, USA
SOURCE: AAAS Annual Meeting and Science Innovation Exposition, (14-19 February, 2002) Vol. 168, pp. A72. print.
Meeting Info.: Annual Meeting of the American Association for the Advancement of Science. Boston, MA, USA. February 14-19, 2002.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jul 2002
Last Updated on STN: 23 Sep 2002

L2 ANSWER 7 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI New LIM2 inhibitor of LMO2, useful in preparing a medicament for the prophylaxis and/or treatment of a condition, e.g. tumor formation, tumor metastasis, inflammation, LMO2 mediated T-cell leukemia or diabetic retinopathy.
AN 2005-333431 [34] WPIDS
AB WO2005039613 A UPAB: 20050527
NOVELTY - An LIM2 inhibitor which is capable of binding to the LIM2 domain

of LMO2 (LIM Domain only 2) and inhibiting the functional activity of LMO2, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) nucleic acid encoding an anti-LMO2 antibody (LIM2 inhibitor) described above;

(2) a vector comprising the nucleic acid;

(3) a host cell comprising the vector;

(4) a composition comprising one or more LIM2 inhibitors described above and a pharmaceutical carrier, diluent or excipient; and

(5) a method for the prophylaxis and/or treatment of any one or more conditions comprising administration to an individual the one or more LIM2 inhibitors described above.

ACTIVITY - Cytostatic; Antiinflammatory; Antidiabetic; Ophthalmological.

No biological data given.

MECHANISM OF ACTION - None given.

USE - The LIM2 inhibitor or composition is useful for inhibiting the functional activity of LMO2 or in preparing a medicament for inhibiting the functional activity of LMO2 or a medicament for the prophylaxis and/or treatment of one or more conditions, e.g. tumor formation, tumor metastasis, inflammation, LMO2 mediated T-cell leukemia or diabetic retinopathy or is used in medicine (claimed).

Dwg.0/8

ACCESSION NUMBER: 2005-333431 [34] WPIDS
DOC. NO. CPI: C2005-103669
TITLE: New LIM2 inhibitor of LMO2, useful in preparing a medicament for the prophylaxis and/or treatment of a condition, e.g. tumor formation, tumor metastasis, inflammation, LMO2 mediated T-cell leukemia or diabetic retinopathy.
DERWENT CLASS: B04 D16
INVENTOR(S): APPERT, A; TERENCE, H R
PATENT ASSIGNEE(S): (MEDI-N) MEDICAL RES COUNCIL
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2005039613	A1	20050506	(200534)*	EN	77

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005039613	A1	WO 2004-GB4299	20041008

PRIORITY APPLN. INFO: GB 2003-24265 20031016

L2 ANSWER 8 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI Identifying peptide aptamers capable of altering cell phenotype comprises screening cells with a nucleic acid library is useful for finding new peptides to treat associated disease such as cancer or osteoporosis.
AN 2001-662979 [76] WPIDS
AB WO 200175178 A UPAB: 20011227

NOVELTY - Identifying, (M1), a **peptide aptamer** capable

of modifying a cell phenotype, comprising contacting cells with a library of nucleic acids encoding random peptide aptamers, selecting a cell with an altered phenotype and identifying the aptamer(s) expressed in the cell, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a **peptide aptamer** or its derivative identified by M1;

(2) treating a disease or condition associated with an aberrant cell phenotype, comprising administering a **peptide aptamer** or its derivative identified by M1;

(3) a viral vector encoding a **peptide aptamer** suitable for treating a disease characterized by an aberrant cell phenotype.

ACTIVITY - Cytostatic; osteopathic; circulatory.

MECHANISM OF ACTION - Gene therapy. No details are given.

USE - Identified aptamers are used to treat a disease or condition associated with an aberrant cell phenotype, especially altered of apoptosis, signal transduction, protein trafficking, cell adhesion, membrane transport, cell motility or differentiation. Particular diseases are cancer, osteoporosis or hematochromatosis.

Dwg.0/5

ACCESSION NUMBER: 2001-662979 [76] WPIDS
DOC. NO. CPI: C2001-194771
TITLE: Identifying peptide aptamers capable of altering cell phenotype comprises screening cells with a nucleic acid library is useful for finding new peptides to treat associated disease such as cancer or osteoporosis.
DERWENT CLASS: B04 D16
INVENTOR(S): BENSON, J D; BRASHER, B B; VINCENT, S M
PATENT ASSIGNEE(S): (ENAN-N) ENANTA PHARM INC
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001075178	A2	20011011	(200176)*	EN	21
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001089284	A	20011015	(200209)		
US 2003108532	A1	20030612	(200340)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001075178	A2	WO 2001-US10953	20010404
AU 2001089284	A	AU 2001-89284	20010404
US 2003108532	A1 Provisional	US 2000-194722P	20000404
	Cont of	WO 2001-US10953	20010404
		US 2002-263577	20021003

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001089284	A Based on	WO 2001075178

PRIORITY APPLN. INFO: US 2000-194722P 20000404; US

L2 ANSWER 9 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI New peptides that bind hepatitis human papilloma virus E6 protein, useful for treatment and diagnosis of infection and associated diseases, also related nucleic acid and antibodies.
AN 2000-515461 [47] WPIDS
AB DE 19901008 A UPAB: 20000925
NOVELTY - Peptides (I), and their variants with up to 40% modification of their sequences, are new.

DETAILED DESCRIPTION - Peptides (I) have the formulae:
NH2-GALVHKLFSQTSGSCLVCIS-COOH NH2-LDVLGCLVRRLGVVLVGLH-COOH
NH2-CYVECGCEVLTALVNGVRVL-COOH NH2-GVGGLCSCASCVSEDFYASV-COOH
NH2-IDLLRRLSQLHLLLVSVGG-COOH NH2-LAVLLNGYTRAIVGISFGGW-COOH
NH2-LCTMCATVFRPLLVWFWSIW-COOH NH2-QLLLDLLLGSYEGMSLTSSP-COOH
NH2-SRSNALHTLDVLLGGT-COOH NH2-GGAVYLCDAGCCFYCCGCSG-COOH
NH2-CLELFDDLFLALSLLLLVGG-COOH NH2-PLCRTCLIESAVLIQLSRL-COOH
NH2-VFSGVYYAEFVFAASAGGTP-COOH NH2-MAPVGAGRPCCTVCFLTARF-COOH
NH2-LSMLLFAAKLPVAVLCSWQA-COOH NH2-LVGRVRIGVSVFIRGGRLL-COOH
NH2-LFDIFRLCAQPVLVHGHTRV-COOH. INDEPENDENT CLAIMS are also included for the following:

(1) DNA (II) that encodes (I);
(2) an expression vector that contains (II);
(3) antibodies (Ab) directed against (I); and
(4) a composition comprising at least one of (I), vectors of (2) and/or Ab, plus usual auxiliaries.

ACTIVITY - Antiviral; anticancer.

MECHANISM OF ACTION - (I) bind to HPV E6 protein, so inhibit its anti-apoptotic activity, resulting in elimination of HPV-positive cells. Vector pCEP4 was modified to express peptide NH2-GALVHKLFSQTSGSCLVCIS-COOH as a fusion with the herpes simplex virus-1 VP22 protein. The vector was used to transfect cervical carcinoma cells and the morphology and growth of the cells monitored. Analysis showed inhibition of both the anti-apoptotic activity of E6 and growth of the cells.

USE - (I), also vectors containing DNA that encodes (I) and/or antibodies directed against (I), are used to treat human papilloma virus (HPV) infections and associated diseases, especially dysplasia and carcinoma, specifically of the cervix uteri. Also (not claimed), (I) are used (i) to detect HBV core proteins for diagnosis of disease and (ii) to detect Ab. Ab are useful for monitoring treatment of the specified diseases.

DESCRIPTION OF DRAWING(S) - Scheme showing **peptide-aptamer** screening in yeast. If E6 protein binds to randomized 20-mer peptide, presented within **thioredoxin**, then the selection gene (ADE2) will be activated.

Dwg.1/2

ACCESSION NUMBER: 2000-515461 [47] WPIDS
DOC. NO. CPI: C2000-153862
TITLE: New peptides that bind hepatitis human papilloma virus E6 protein, useful for treatment and diagnosis of infection and associated diseases, also related nucleic acid and antibodies.
DERWENT CLASS: B04 D16
INVENTOR(S): BUTZ, K; HOPPE-SEYLER, F
PATENT ASSIGNEE(S): (DEKR-N) DEUT KREBSFORSCHUNGSZENTRUM
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
DE 19901008	A1	20000720	(200047)*		7
WO 2000042064	A1	20000720	(200047)	GE	

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE

W: JP US
EP 1140987 A1 20011010 (200167) GE
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 2002535969 W 20021029 (200274) 24
EP 1140987 B1 20030514 (200333) GE
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
DE 50002164 G 20030618 (200341)
US 6610473 B1 20030826 (200357)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
DE 19901008	A1	DE 1999-1001008	19990113
WO 2000042064	A1	WO 2000-DE141	20000112
EP 1140987	A1	EP 2000-908931	20000112
		WO 2000-DE141	20000112
JP 2002535969	W	JP 2000-593631	20000112
		WO 2000-DE141	20000112
EP 1140987	B1	EP 2000-908931	20000112
		WO 2000-DE141	20000112
DE 50002164	G	DE 2000-00002164	20000112
		EP 2000-908931	20000112
		WO 2000-DE141	20000112
US 6610473	B1	WO 2000-DE141	20000112
		US 2001-889136	20011004

FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 1140987	A1 Based on	WO 2000042064
JP 2002535969	W Based on	WO 2000042064
EP 1140987	B1 Based on	WO 2000042064
DE 50002164	G Based on	EP 1140987
	Based on	WO 2000042064
US 6610473	B1 Based on	WO 2000042064

PRIORITY APPLN. INFO: DE 1999-19901008 19990113

L2 ANSWER 10 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI New peptides that bind hepatitis B core protein, useful for treatment and diagnosis of hepatitis B infection and associated diseases.
AN 2000-499838 [45] WPIDS
AB DE 19901009 A UPAB: 20000918
NOVELTY - Peptides (I), and their variants with up to 40% modification of their sequences, are new.
DETAILED DESCRIPTION - Peptides (I) have the formulae:
NH2-SFYSVLFLWGTCGGFSHSWY-COOH;
NH2-LCETVRWPVCFCSLYVICS-COOH;
NH2-SCAPAWSPAPTVVFVALYVV-COOH;
NH2-QWGMDSLIRLYLWESLGLLS-COOH;
NH2-IHPLSRGNFFPHVRLMGEWR-COOH;
NH2-GQALCAGVSLFADWLIIESTL-COOH;
NH2-LKHFDPRWPLMSLMSSWACM-COOH;
NH2-PPLRKAFCWRCFNWLSTKRL-COOH; and
NH2-LRKSMLKVGRDVCYVSLWVF-COOH. INDEPENDENT CLAIMS are also included for the following:
(1) a DNA (II) that encodes (I);
(2) an expression vector that contains (II);
(3) antibodies (III) directed against (I); and
(4) a composition comprising at least one of (I), (II) and/or (III).
ACTIVITY - Antiviral; anticancer.

MECHANISM OF ACTION - (I) bind to HBV core proteins, so inhibit viral replication. Vector pCEP4 was modified to express peptide C1 NH2-SFYSVLFLWGTCGGFSHSWY-COOH (C1) as a fusion with the herpes simplex virus-1 VP22 protein, forming pCEP4-C1-1. This was used to transfect HepG2 hepatoma cells together with an expression plasmid encoding HBV. Cells and culture supernatant were analyzed for HBV particles and nucleic acids and the results indicated strong inhibition of particle/nucleic acid production. Control cells that did not express C1 did not show any inhibition.

USE - The peptides, DNA molecules and/or antibodies are used to treat hepatitis B virus (HBV) infections and associated diseases, especially chronic hepatitis and hepatocellular carcinoma. The peptides are used

(i) to detect HBV core proteins for diagnosis of disease; and

(ii) to detect Ab. Ab are useful for monitoring treatment of the specified diseases.

DESCRIPTION OF DRAWING(S) - Scheme showing **peptide-aptamer** screening in yeast. If the core protein (C) binds to a randomized 20-mer peptide, presented within **thioredoxin**, then the selection gene (ADE2) will be activated.

Dwg.1/2

ACCESSION NUMBER: 2000-499838 [45] WPIDS
DOC. NO. CPI: C2000-150139
TITLE: New peptides that bind hepatitis B core protein, useful for treatment and diagnosis of hepatitis B infection and associated diseases.
DERWENT CLASS: B04 D16
INVENTOR(S): BUTZ, K; HOPPE-SEYLER, F
PATENT ASSIGNEE(S): (DEKR-N) DEUT KREBSFORSCHUNGSZENTRUM
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
DE 19901009	A1	20000720	(200045)*		7
WO 2000042063	A2	20000720	(200045)	GE	
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: JP US					
EP 1140995	A2	20011010	(200167)	GE	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2002534111	W	20021015	(200282)		19

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
DE 19901009	A1	DE 1999-1001009	19990113
WO 2000042063	A2	WO 2000-DE140	20000112
EP 1140995	A2	EP 2000-908930	20000112
		WO 2000-DE140	20000112
JP 2002534111	W	JP 2000-593630	20000112
		WO 2000-DE140	20000112

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 1140995	A2	Based on	WO 2000042063
JP 2002534111	W	Based on	WO 2000042063

PRIORITY APPLN. INFO: DE 1999-19901009 19990113

=> s intracellular recognition molecule

L3 1 INTRACELLULAR RECOGNITION MOLECULE

=> d l3 ti abs ibib tot

L3 ANSWER 1 OF 1 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI Process for specifically modulating the properties of an intracellular target molecule used for the treatment of various disorders.
AN 2002-418829 [45] WPIDS
AB EP 1205191 A UPAB: 20020717
NOVELTY - Process for specifically modulating the properties of an intracellular target molecule T, and/or of a cellular component C which interacts directly or indirectly in a cell with T.

DETAILED DESCRIPTION - Process for specifically modulating the properties of an intracellular target molecule T, and/or of a cellular component C which interacts directly or indirectly in a cell with T, comprising:

(a) introducing into a cell a chimeric molecule, a so-called targeted effector, comprising:

(i) a recognition moiety R having the capacity to specifically interact within the cell, with a site on an intracellular target molecule T, R interacting with T with a first affinity A1; and

(ii) an effector moiety, E covalently linked to the recognition moiety R, E being a molecule or portion which has an initial capacity to exert an effect on at least one molecule M, and which when it is covalently linked to R, acquires the capacity to specifically exert on the intracellular target molecule, T.

INDEPENDENT CLAIMS are also included for the following:

(1) process for the production of a targeted effector having the capacity to specifically modulate the properties of an intracellular target molecule T, and/or a cellular component C which interacts directly or indirectly in a cell with T comprising:

(i) production of a random pool of peptides, so called recognition moieties R;

(ii) screening of the random pool produced in (i) against T in a cell, in conditions suitable to allow identification of recognition moieties R capable of interacting with T;

(iii) optionally contacting the moieties selected in (ii) with proteins other than T to determine the specificity range of each of said moieties, and to identify moieties having a desired specificity range;

(iv) covalent linkage of the recognition moieties R to an effector moiety E, E being a molecule which initially has the capacity to exert a predetermined effect on at least one intracellular component M.;

(v) verification of the affinity A1 with which the recognition moiety R interacts with T, or of the affinity A2 with which the targeted effector, interacts with T;

(vi) if both of A1 and A2 correspond to Kd values greater than 1 x 10-8M, alteration of the binding region of the effector moiety to adjust the binding affinity of the interaction between T and the selected moiety so that the Kd becomes less than 1 x 10-8M;

(2) process for conferring on an effector moiety E the ability to specifically modulate the properties of an intracellular protein T, or an intracellular component which interacts directly or indirectly with T, comprising:

(i) covalently linking the effector moiety E to a recognition moiety R where R comprises a molecule having the capacity to specifically interact within a cell with a site on an intracellular target molecule T, the interaction with T occurring with an affinity A1 which corresponds to a Kd value of less than 1 x 10-8M and E being a molecule which has an initial capacity to exert the effect on the intracellular target molecule T; and

(ii) optionally optimizing the affinity of the interaction between T and R by altering the chemical composition of the binding region of R to provide an affinity in the desired range;

(3) chimeric molecule, so called targeted effector comprising:

(i) a recognition moiety R having the capacity to specifically interact within a cell with a site on an intracellular target molecule T the interaction with T occurring with an affinity A1; and

(ii) an effector moiety E, covalently linked to R, E being a molecule which has an initial capacity to exert an effect on at least one molecule M, and which when it is covalently linked to R, acquires the capacity to specifically exert the effect on the intracellular target molecule T;

(4) nucleic acid encoding a chimeric protein operably linked to regulatory sequences for expression in a eukaryotic cell;

(5) vector capable of stably introducing a nucleic acid into a prokaryotic or eukaryotic cell;

(6) pharmaceutical composition comprising a chimeric molecule, or a nucleic acid in association with a pharmaceutically acceptable excipient; and

(7) an **intracellular recognition molecule** R, composed of a conformationally constrained recognition domain, displayed in a platform.

ACTIVITY - Antimicrobial; Immunomodulatory; Nootropic; Neuroprotective; Metabolic; Neuroleptic; Cytostatic; Cardiant.

MECHANISM OF ACTION - None given in the specification.

USE - The chimeric protein or nucleic acid is used in the preparation of a medicament for the treatment of microbial infections, immunological disorders, neurological disorders, metabolic disorders, psychiatric disorders, myopathies, genetic disorders, cancer, cardiovascular disorders and dental disorders (claimed).

Dwg.0/7

ACCESSION NUMBER: 2002-418829 [45] WPIDS
DOC. NO. CPI: C2002-118325
TITLE: Process for specifically modulating the properties of an intracellular target molecule used for the treatment of various disorders.
DERWENT CLASS: B04 D16
INVENTOR(S): BRENT, R; COHEN, B A; COLAS, P
PATENT ASSIGNEE(S): (CNRS) CENT NAT RECH SCI; (MASS-N) MASSACHUSETTS GEN HOSPITAL; (MOLE-N) MOLECULAR SCI INST; (BREN-I) BRENT R; (COHE-I) COHEN B A; (COLA-I) COLAS P
COUNTRY COUNT: 99
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1205191	A1	20020515	(200245)*	EN	33
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
WO 2002055108	A1	20020718	(200257)	EN	
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
US 2003143626	A1	20030731	(200354)		
EP 1345627	A1	20030924	(200363)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
AU 2002219153	A1	20020724	(200427)		
JP 2004516848	W	20040610	(200438)		254

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

EP 1205191	A1	EP 2000-403156	20001113
WO 2002055108	A1	WO 2001-EP14199	20011113
US 2003143626	A1	US 2001-66965	20011113
EP 1345627	A1	EP 2001-273076	20011113
		WO 2001-EP14199	20011113
AU 2002219153	A1	AU 2002-219153	20011113
JP 2004516848	W	WO 2001-EP14199	20011113
		JP 2002-555840	20011113

FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 1345627	A1 Based on	WO 2002055108
AU 2002219153	A1 Based on	WO 2002055108
JP 2004516848	W Based on	WO 2002055108

PRIORITY APPLN. INFO: EP 2000-403156 20001113

=> d his

(FILE 'HOME' ENTERED AT 14:46:37 ON 13 MAY 2006)

FILE 'MEDLINE, BIOSIS, WPIDS, DGENE' ENTERED AT 14:46:58 ON 13 MAY 2006
L1 311 S PEPTIDE APTAMER
L2 10 S L1 AND THIOREDOXIN
L3 1 S INTRACELLULAR RECOGNITION MOLECULE

=> s l1 and (conformationally constrained)
L4 1 L1 AND (CONFORMATIONALLY CONSTRAINED)

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Targeted modification and transportation of cellular proteins.
AB Peptide aptamers are proteins selected from combinatorial libraries that display **conformationally constrained** variable regions.
Peptide aptamers can disrupt specific protein interactions and thus represent a useful method for manipulating protein function in vivo. Here, we describe aptamer derivatives that extend the range of functional manipulations. We isolated an aptamer with increased affinity for its Cdk2 target by mutagenizing an existing aptamer and identifying tighter binding mutants with calibrated two-hybrid reporter genes. We used this and other anti-Cdk2 aptamers as recognition domains in chimeric proteins that contained other functional moieties. Aptamers fused to the catalytic domain of a ubiquitin ligase specifically decorated LexA-Cdk2 with ubiquitin moieties in vivo. Aptamers against Cdk2 and another protein, Ste5, that carried a nuclear localization sequence transported their targets into the nucleus. These experiments indicate that fusion proteins containing aptameric recognition moieties will be useful for specific modification of protein function in vivo.
ACCESSION NUMBER: 2001:60196 BIOSIS
DOCUMENT NUMBER: PREV200100060196
TITLE: Targeted modification and transportation of cellular proteins.
AUTHOR(S): Colas, Pierre; Cohen, Barak; Ferrigno, Paul Ko; Silver, Pamela A.; Brent, Roger [Reprint author]
CORPORATE SOURCE: The Molecular Sciences Institute, Berkeley, CA, 94704, USA brent@molsci.org
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (December 5, 2000) Vol. 97, No. 25, pp. 13720-13725. print.

CODEN: PNASA6. ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jan 2001
Last Updated on STN: 12 Feb 2002

=> s l1 and covalent bond
L5 1 L1 AND COVALENT BOND

=> d l5 ti abs ibib tot

L5 ANSWER 1 OF 1 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI Producing fusion molecule capable of use as detector molecule for binding predetermined target analyte by attaching reactive group to protein, bonding coupling reagent, catalyzing reaction between group and reagent.
AN 2004-041346 [04] WPIDS
AB US2003198973 A UPAB: 20040115
NOVELTY - Producing a fusion molecule by attaching a reactive group to an end of a protein sub-unit, bonding a coupling reagent to an end of a nucleic acid, the coupling reagent of the modified nucleic acid capable of displacing the reactive group of the reactive intermediate and catalyzing a reaction between the reactive group and coupling reagent.

DETAILED DESCRIPTION - Producing (M1) a fusion molecule for use as a detector molecule for binding a predetermined target analyte, comprising attaching a reactive group to an end of a protein sub-unit, creating a reactive intermediate with the reactive group at an end, bonding a coupling reagent to an end of a nucleic acid, forming a modified nucleic acid, the coupling reagent of the modified nucleic acid being capable of displacing the reactive group of the reactive intermediate and catalyzing a reaction between the reactive group of the reactive intermediate and the coupling reagent of the modified nucleic acid, where in the reaction, the reactive group is displaced from the end of the reactive intermediate and a **covalent bond** is formed between the end of the reactive intermediate and the end of the modified nucleic acid. The method optionally (M2) involves attaching a reactive group to an end of a protein sub-unit, creating a reactive intermediate with the reactive group at its end, bonding a phosphoramidite-containing molecule to an end of a nucleic acid, forming a modified nucleic acid, the phosphoramidite-containing molecule of the modified nucleic acid being capable of displacing the reactive group of the reactive intermediate and catalyzing a reaction between the reactive group of the reactive intermediate and the phosphoramidite-containing molecule of the modified nucleic acid, where in the reaction, the reactive group is displaced from the end of the reactive intermediate and a **covalent bond** is formed between the end of the reactive intermediate and the end of the modified nucleic acid. The method optionally (M3) involves attaching a reactive group to an end of a protein sub-unit, creating a reactive intermediate with the reactive group at an end, attaching a coupling reagent to a nucleotide, forming a modified nucleotide, linking the modified nucleotide to an end of a nucleic acid, forming a modified nucleic acid, the coupling reagent of the modified nucleic acid being capable of displacing the reactive group of the reactive intermediate, and catalyzing a reaction between the reactive group of the reactive intermediate and the coupling reagent of the modified nucleic acid. The reactive group is displaced from the end of the reactive intermediate and a **covalent bond** is formed between an end of the reactive intermediate and the end of the modified nucleic acid. The method optionally (M4) involves attaching a reactive group to an end of a protein sub-unit, creating a reactive intermediate with the reactive group at an end, attaching a cysteine-like group to a nucleotide forming a modified nucleotide, linking an end of a nucleic acid to the modified nucleotide, forming a modified nucleic acid, the cysteine-like group of the modified nucleic acid being capable of

displacing the reactive group of the reactive intermediate and catalyzing a reaction between the reactive group of the reactive intermediate and the cysteine-like group of the modified nucleic acid , where in the reaction, the reactive group is displaced from the end of the reactive intermediate and a **covalent bond** is formed between the end of the reactive intermediate and the end of the modified nucleic acid. The method optionally (M5) involves attaching reactive groups to ends of protein sub-units of a quantity of protein sub-units, creating a quantity of reactive intermediates with the reactive groups at ends, bonding coupling reagents to second nucleotides, forming second modified nucleotides, linking ends of a quantity of nucleic acids to the modified nucleotides, and linking second ends of the quantity of the nucleic acids to the second modified nucleotides, forming modified nucleic acids with first and seconds ends, severing the modified nucleic acids between the first and the second ends, thereby forming, from the first end from the modified nucleic acid modified nucleic acid fragments containing the first modified nucleotide and from the second end from the modified nucleic acid modified nucleic acid fragments containing the second modified nucleotide and catalyzing a first reaction between the first coupling reagent of the first modified nucleic acid fragments and reactive groups of the reactive intermediates of the quantity , wherein in the reaction, the reactive groups are displaced from the first ends of the reactive intermediates, and second covalent bonds are formed between the reactive intermediates and the second modified nucleotides of the second modified nucleic acid fragments.

INDEPENDENT CLAIMS are included for the following:

(1) a fusion molecule (I) capable of binding a predetermined target analyte comprising a protein sub-unit, a linker attached to an end of the protein sub-unit and a DNA molecule attached at an end to the linker by a **covalent bond** or comprising a protein sub unit a cysteine-like group attached to a first end of the protein sub-unit and a nucleic acid attached at an end to the cysteine-like group by a **covalent bond**;

(2) product (II) of (M1);

(3) product (III) of (M5); and

(4) a kit (IV) for use in recognizing or quantifying a target analyte, comprising a detector fusion molecule capable of binding to a target analyte, the detector fusion molecule which comprises a protein sub-unit, a linker attached to an end of the protein sub-unit and a DNA attached at an end to the linker by a **covalent bond**, a unit for amplifying the detector fusion molecule, producing an amplification product, and a unit for visualizing the amplification product.

USE - (I) is useful for recognizing a target analyte in a sample. (I) is useful for quantifying target analyte in a sample. (M3) further comprises resolving the amplification product on a basis of size. (I) is useful for creating a nanostructure on a target analyte (all claimed).

Dwg.0/25

ACCESSION NUMBER: 2004-041346 [04] WPIDS
DOC. NO. NON-CPI: N2004-033481
DOC. NO. CPI: C2004-016763
TITLE: Producing fusion molecule capable of use as detector molecule for binding predetermined target analyte by attaching reactive group to protein, bonding coupling reagent, catalyzing reaction between group and reagent.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BURBULIS, I E; CARLSON, R H
PATENT ASSIGNEE(S): (MOLE-N) MOLECULAR SCI INST INC; (MOLE-N) MOLECULAR SCI INST
COUNTRY COUNT: 104
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

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US 2003198973   A1 20031023 (200404)*      41
WO 2003091404   A2 20031106 (200404)  EN
   RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS
       LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
    W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
       DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
       KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL
       PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU
       ZA ZM ZW
AU 2003231760   A1 20031110 (200442)
EP 1499747      A2 20050126 (200508)  EN
    R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
       MC MK NL PT RO SE SI SK TR
JP 2005523699   W  20050811 (200554)       32
AU 2003231760   A8 20051020 (200615)
US 2006073481   A1 20060406 (200625)#
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APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2003198973	A1 Provisional	US 2002-374795P	20020423
		US 2002-218233	20020812
WO 2003091404	A2	WO 2003-US12797	20030423
AU 2003231760	A1	AU 2003-231760	20030423
EP 1499747	A2	EP 2003-747316	20030423
		WO 2003-US12797	20030423
JP 2005523699	W	JP 2003-587940	20030423
		WO 2003-US12797	20030423
AU 2003231760	A8	AU 2003-231760	20030423
US 2006073481	A1 Provisional	US 2002-374795P	20020423
		WO 2003-US12797	20030423
		US 2004-515108	20041119

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003231760	A1 Based on	WO 2003091404
EP 1499747	A2 Based on	WO 2003091404
JP 2005523699	W Based on	WO 2003091404
AU 2003231760	A8 Based on	WO 2003091404

PRIORITY APPLN. INFO: US 2002-374795P 20020423; US
2002-218233 20020812; US
2004-515108 20041119